and at which time the Examiner noted that complete copies of any of the U.S. Patents mentioned in the April 1, 2002 amendment (discussed below) did not have to be submitted by applicant as she could obtain such copies herself if needed.

Claims 1-5 are drawn to the elected method of treating a Th2-dominated disease (claims 1, 4 and 5), inhibiting a pathogenic Th2 response (claim 2), or stimulating the development of activated human T cells into Th1-like effectors (claim 3).

Claim 6 is drawn to the non-elected method of regulating immune homeostasis during pregnancy.

Per the April 1, 2002 amendment, a computer readable form (CRF) and associated paper copy sequence have been requested to be entered, to which no new matter has been added. As the Examiner has withdrawn the criticism of failure to comply with the sequence rules 37 CFR 1.821-825 in not referring in the disclosure to the required sequence identifier (SEQ ID NO:), i.e., in view of the specification revision per the August 30, 2001 amendment, it is believed that all criticisms of failure to comply with such sequence rules are now overcome.

I - INDEFINITENESS REJECTION

Reconsideration is requested of the rejection of the claims under 35 USC 112, second paragraph, as indefinite in not defining in the specification what is encompassed by the terms "Th2-dominated disease" (claims 1, 3,4 and 5) and "pathogenic Th2 response" (claim 2).

The Examiner notes that there is evidence, i.e., in Bigazzi US-296 which is used in the double patenting rejection discussed below, that Th2-dominated diseases are contemplated therein, but that there is no evidence of what is encompassed by the terms "Th2-dominated disease" and "pathogenic Th2 response," and hence the metes and bounds of the claims cannot be determined.

The April 1, 2002 amendment included copies of:

the three articles:

[1] Romagnani, Int J Clin Lab Res 1996, 26:83-98 ("Romagnani I"),

[2] Grunewald et al., J Immunol 1998, 160:4004-4009 ("Grunewald"), and

[3] Romagnani, Ann Allergy Asthma Immunol 2000, 85:9-21 ("Romagnani II"),

plus:

[4] an internet version partial copy of U.S. Patent No. 6,086,898, issued July 11, 2000 to DeKruyff et al., but hereinafter all references thereto will be in terms of the column and line official version ("[4] DeKruyff US-898"),

[5] cover page only of U.S. Patent No. 6,066,322, issued May 23, 2000 to Levinson, but hereinafter all references thereto will be in terms of the complete copy thereof ("[5] Levinson US-322"),

[6] cover page only of U.S. Patent No. 6,156,887, issued December 5, 2000 to Levinson, but hereinafter all references thereto will be in terms of the complete copy thereof ("[6] Levinson US-887"),

[7] cover page only of U.S. Patent No. 6,190,909, issued February 20, 2001 to Levinson et al., but hereinafter all references thereto will be in terms of the complete copy thereof ("[7] Levinson US-909"),

[8] cover page only of U.S. Patent No. 6,288,218, issued September 11, 2001 to Levinson, but hereinafter all references thereto will be in terms of the complete copy thereof ("[8] Levinson US-218"),

[9] cover page only of U.S. Patent No. 6,323,334, issued November 27, 2001 to Kingsbury et al., but hereinafter all references thereto will be in terms of the complete copy thereof ("[9] Kingsbury US-334"),

as well as:

[10] cover page only of U.S. Patent No. 5,721,351, issued February 24, 1998 to Levinson, ("[10] Levinson US-351"),

[11] cover page only of U.S. Patent No. 6,066,498, issued May 23, 2000 to Levinson ("[11] Levinson US-498"),

[12] cover page only of U.S. Patent No. 6,084,083, issued July 4, 2000 to Levinson ("[12] Levinson US-083"), and

[13] cover page only of U.S. Patent No. 6,204,371, issued March 20, 2001 to Levinson ("[13] Levinson US-371").

It is noted that [10] Levinson US-351 is a continuation in part (CIP) of [5] Levinson US-322; that [11] Levinson US-498 is a division of [10] Levinson US-351 and in turn a CIP of [5] Levinson US-322; that [12] Levinson US-083 is a division of [13] Levinson US-371 which is a CIP of [10] Levinson US-351 and in turn of [5]

Levinson US-322; and that [13] Levinson US-371 is a CIP of [10] Levinson US-351 and in turn of [5] Levinson US-322.

Hence, such patents [10] to [13] may be deemed to duplicate generally the content of the earlier mentioned patents [5] to [9]. Accordingly, only the articles [1] to [3] and patents [4] to [9] are discussed below, with patents [10] to [13] being regarded as cumulative thereto.

[1] Romagnani I (as of 1996) discusses the development of Th1- and Th2-dominated responses, noting that type 1 helper T cells (Th1) and type 2 helper T cells (Th2) are polarized forms of the effector specific immune response, based on cytokine production, and play different roles in protection and immunopathology, involving many factors that regulate type 1 and type 2 helper cell development in response to antigen stimulation (Abstract).

In this regard (p. 83), cellular and humoral arms of the specific immune response are regulated by distinct subsets of $CD4^+$ Th1 and Th2 cells, respectively. Th cells respond to antigenic stimulation with cytokines:

Th1 cells secrete interleukin-2 (IL-2), tumor necrosis factor-β (TNF-β) and interferon-γ, and due to the macrophage activating property of the latter Th1 cells are the principal effectors of cell mediated immunity (CMI) against intracellular microbes and delayed type hypersensitivity (DTH) reactions; Th1 cells also stimulate production of antibodies effective at activating complement and opsonizing antigens for phagocytosis, and

thus trigger phagocyte mediated host defense, and infection with intracellular microbes tends to induce Th1 type response;

Th2 cells produce IL-4, IL-13 (in turn stimulating IgE production), IL-3, IL-10 (which with IL-4 act as mast cell growth factors), and IL-5 (an eosinophil activating factor) for protection against helminthic (e.g., intestinal worm) infections; by producing IL-4, IL-10 and IL-13, Th2 cells can inhibit macrophage activity.

A suggested Th2 cell function is to ameliorate the tissue damaging effect of protective immune response which occurs when Th1 cells respond to an intracellular infectious agent, yet since Th2 cells produce IL-4 and IL-5, and react with common environmental allergens, they are involved in pathogenesis of allergic IgE mediated reactions.

Absent clear polarizing signals, CD4+ Th cell subsets with a less differentiated cytokine profile than Th1 or Th2 cells, designated Th0, usually arise, which may dominate the earliest stages of some responses and mediate intermediate effector functions.

Fig. 1 of [1] Romagnani I shows (p. 84) the role of Th1 and Th2 cells in the effector response against infectious agents, and it is noted that infectious agents evoke Th1 type responses which, per production of opsonizing and complement fixing antibodies by B cells and activation of phagocytic cells, eliminate the microbes. On the other hand, if the microbes are not removed, the persisting Th1 response results in inflammatory tissue injury. Frequently, a

switch from Th1 to Th2 dominated response occurs, which controls inflammation.

Parenthetically, it is noted (p. 84) that Table 1 of [1] Romagnani I treats factors involved in differentiation of $CD4^+$ T cells into Th1 or Th2 phenotype, including, inter alia, cytokines, hormones such as glucocorticoids, androgen steroids, 25 (OH) D_3, progesterone and relaxin, and (p. 92, col. 2, bottom half) that, unlike progesterone, relaxin favors development of IFN-γ and TNF-β producing cells without influencing IL-4 and IL-5 production.

[1] Romagnani I notes that (p.85, col. 2, bottom half) autoimmune encephalomyelitis is a Th1 mediated disorder and nonobese diabetes is also a Th1 dependent autoimmune disorder, that (p. 86, col. 1, top half) colitis is a Th1 mediated disease, that (p. 87, col. 2, bottom half) the cytokine IL-12 is the dominant factor in directing the development of Th1 cells [see also Fig. 2 (p. 88, col. 1) where it is also indicated that helminths and environmental allergens promote Th2 cell development by production of IL-4]; and that Th2 development is also induced by hormonal, i.e., progesterone, influence], that (p. 91, col. 1, top half) leprosy is a Th1 dominated disease, that (p. 93, col. 1, bottom half) intracellular pathogens promote Th1 dominated responses while extracellular pathogens, soluble antigens and oral immunization promote Th2 dominated responses.

[1] Romagnani I concludes (p. 93-94) that Th1 cells are responsible for cell-mediated inflammatory reactions per involvement of phagocytic cells, while Th2 cytokines promote antibody

production, enhance eosinophil differentiation and function, and inhibit macrophage functions, with Th2 cells being associated with antibody and allergic responses and protecting against tissue damaging effects of Th1 mediated immune reactions; Th1 and Th2 cells are effector cells originating from common precursors consequent repeated antigenic stimulation.

[2] Grunewald (as of 1998) is concerned with the effects of an antagonistic mutant IL-4 on such Th2 dominated diseases as immediate cutaneous hypersensitivity and anaphylactic shock, and its inhibition of humoral immune response and allergic reactivity (Abstract), noting the role of IL-4 in Th2 dominated diseases like type I allergies, helminthic infections and autoimmune diseases such as systemic sclerosis, systemic lupis erythematosus (p. 4004, col. 1).

[3] Romagnani II (as of 2000) is a review article as to Th1 versus Th2 T cell subsets in polarized specific immune responses mediated by $CD4^+$ Th lymphocytes based on cytokine production profiles, i.e., of Th1 or Th2 type cells, which concludes (p. 9) that:

Th1 cells, which produce interferon-γ (IFN-γ), interleukin-2 (IL-2) and tumor necrosis factor-β (TNF-β), evoke cell mediated immunity and phagocyte dependant inflammation,

Th2 cells, which produce IL-4, IL-5, IL-6, IL-9, IL-10 and IL-13, evoke antibody responses (including IgE responses) and

eosinophil accumulation, but inhibit phagocyte independent inflammation,

Th1 dominated responses are involved in the pathogenesis of organ specific autoimmune disorders, Crohn's disease, sarcoidosis, acute kidney allograph rejection, and unexplained recurrent abortions, and

Th2 dominated responses which are allergen specific are responsible for atopic (i.e., allergic) disorders, and Th2 dominated responses play a pathogenic role in progressive systemic sclerosis and cryptogenic fibrosing alveolitis, and favor more rapid HIV infection evolution toward the full blown disease.

[3] Romagnani II notes (p. 12-16, including Tables 3 and 4) Th1 and Th2 responses to specific immunopathologic disorders, and Th1/Th2 paradigm therapeutic implications, e.g., treatment of allergic disorders (including bronchial asthma; Fig. 6) and autoimmune disorders.

[4] DeKruyff US-898 (30 columns with 10 sheets of drawings) shows the immunotherapy of converting a Th2 response, i.e., a TH2 allergic immune response to an antigen, into a Th1 response, i.e., a Th1 immune response, in an individual by administering thereto the antigen plus a Listeria adjuvant. The adjuvant induces a Th1 immune response and suppresses a Th2 allergic immune response, such as to reverse ongoing airway disease or a condition associated with Th2 cytokines and/or IgE antibodies, asthma, allergic rhinitis, or anaphylactic reaction, and convert an allergic inflammatory response into a protective immune response.

[4] DeKruyff US-898 concerns treatment of allergic and other immune disorders associated with overproduction of Th2 type cytokines by antigen specific T cells, such as to inhibit airway hyperactivity and inflammation to reverse ongoing airway disease and convert allergic inflammatory responses into protective immune responses, i.e., using an adjuvant to induce a Th1 type immune response and redirect a Th2 type response to a Th1 type response for a selected antigen, e.g., in treating asthma, allergic rhinitis and anaphylactic reactions (Abstract).

[4] DeKruyff US-898 notes particular allergies (i.e., hypersensitivities) of the (human) immune system, mechanisms of action that occur during Th1 dominated responses and separately during Th2 dominated responses, and specific disorders, e.g., asthma, allergy or atopy, anaphylactic reaction, that involve a Th2 dominated response (col. 1, lines 19-30 and 50-61; col. 1, line 65, to col. 2, line 17; col. 3, lines 14-21; col. 4, lines 8-50; col. 5, lines 1-10 and 25-31; col. 6, lines 33-57; col. 7, lines 9-30 and 50-67; col. 8, lines 31-40; col. 9, lines 1-20; col. 10, lines 10-20; col. 26, lines 26-58; col. 27, lines 31-43 and 55-58; and col. 27, line 66, to col. 28, line 7).

The claims of [4] DeKruyff US-898 confirm that current patent practice permits language (similar to that herein) as to a method of converting an established "Th2-type allergic immune response" to an antigen into a "Th1-type response" whereby to "induce a Th1-type response" and suppress a "Th2-type allergic immune response" (claim

1), for treating asthma (claims 2 and 11), rhino conjunctivitis (claim 3), and anaphylactic reactions (claim 9).

[5] Levinson US-322 (84 columns with 18 sheets of drawings) shows treatment of T lymphocyte related immune disorders, especially Th2 dominated disorders, including chronic inflammatory diseases and disorders such as Crohn's disease, reactive arthritis, including Lyme disease, insulin dependent diabetes, organ specific autoimmunity, including multiple sclerosis, Hashimoto's thyroiditis and Grave's disease, contact dermatitis, psoriasis, graph rejection, graph versus host disease, sarcoidosis, atopic conditions, such as asthma and allergy, including allergic rhinitis, gastrointestinal allergies, including food allergies, eosinophilia, conjunctivitis, glomerular nephritis, certain pathogen susceptibilities, such as helminthic (e.g., leishmaniasis), and certain viral infections, including HIV, and bacterial infections, including tuberculosis and lepromatous leprosy (col. 1, lines 5-24; col. 2, lines 39-43; col. 3, lines 20-52; col. 5, lines 18-30; col. 11, lines 17-23; col. 13, lines 35-40; col. 14, lines 34-47; col. 35, lines 18-35; col. 44, lines 19-62; col. 57, lines 1-2 and 7-10; and col. 58, lines 12-25).

[5] Levinson US-322 notes the two pertinent Th cell types Th1 and Th2 and their functions, etc. (col. 1, lines 41-46 and 53-64; col. 2, lines 11-22 and 31-65; col. 3, lines 10-52; col. 5, lines 13-18; col. 6, lines 50-59; col. 7, lines 15-44; col. 8, lines 9-14; col. 14, lines 17-30; col. 42, lines 42-50; col. 43, lines 50-

56; col. 45, line 55, to col. 56, line 17; col. 50, lines 49-53 and 59-62; col. 51, lines 19-22; and col. 52, lines 29-32 and 61-65).

Like [4] DeKruyff US-898, the claims of [5] Levinson US-322 also confirm that current patent practice permits language (similar to that herein) as to a method of treating asthma to ameliorate its symptoms wherein the asthma is a "Th2 or Th2-like mediated condition" (claim 1), e.g., wherein the asthma is also an IgE-mediated condition (claim 4) or IL-4-mediated condition (claim 6).

[5] Levinson US-887 (136 columns with 37 sheets of drawings), which is a division of [13] Levinson US-371 and in turn a CIP of [10] Levinson US-351 which is itself a CIP of [5] Levinson US-322), insofar as pertinent hereto, similarly to [5] Levinson US-322, shows treatment of T lymphocyte related immune disorders, especially Th2 dominated disorders, including chronic inflammatory diseases and disorders such as Crohn's disease, reactive arthritis, including Lyme disease, insulin dependent diabetes, organ specific autoimmunity, including multiple sclerosis, Hashimoto's thyroiditis and Grave's disease, contact dermatitis, psoriasis, graph rejection, graph versus host disease, sarcoidosis, atopic conditions, such as asthma and allergy, including allergic rhinitis, gastrointestinal allergies, including food allergies, eosinophilia, conjunctivitis, glomerular nephritis, certain pathogen susceptibilities, such as helminthic (e.g., leishmaniasis), and certain viral infections, including HIV, and bacterial infections, including tuberculosis and lepromatous leprosy (col. 1, lines 15-32; col. 2, lines 45-52; col. 3, lines 29-40; col. 5, lines 45-62; col. 13,

line 62, to col. 14, line 1; col. 16, lines 13-19; col. 17, lines 11-26; col. 40, lines 33-51; col. 53, line 49, to col. 54, line 11; and col. 74, lines 25-38).

[6] Levinson US-887 similarly notes the two pertinent Th cell types Th1 and Th2 and their functions, etc. (col. 1, lines 51-55; col. 2, lines 1-4, 20-33 and 41-67; col. 3, lines 1-25 and 48-60; col. 8, lines 45-59; col. 9, lines 5-8 and 19-23; col. 10, lines 9-14; col. 16, line 62, to col. 17, line 10; col. 41, lines 31-34; col. 43, lines 46-54; col. 53, lines 1-5; col. 55, lines 4-34; col. 65, lines 45-49 and 55-58; col. 66, lines 13-15; col. 67, lines 37-39; col. 68, lines 2-6; and col. 72, lines 12-14 and 19-23).

[7] Levinson US-909 (76 columns with 6 sheets of drawings), insofar as pertinent hereto, similarly to [5] Levinson US-322, shows treatment of T lymphocyte related immune disorders, especially Th2 (referred to as STIF) dominated disorders, including chronic inflammatory diseases and disorders such as Crohn's disease, reactive arthritis, including Lyme disease, insulin dependent diabetes, organ specific autoimmunity, including multiple sclerosis, Hashimoto's thyroiditis and Grave's disease, contact dermatitis, psoriasis, graph rejection, graph versus host disease, sarcoidosis, atopic conditions, such as asthma and allergy, including allergic rhinitis, gastrointestinal allergies, including food allergies, eosinophilia, conjunctivitis, glomerular nephritis, certain pathogen susceptibilities, such as helminthic (e.g., leishmaniasis), and certain viral infections, including HIV, and bacterial infections, including tuberculosis and lepromatous

leprosy (col. 1, lines 19-34; col. 3, lines 15-46; col. 4, line 65, to col. 6, line 11; col. 11, lines 37-39; col. 22, lines 26-40; col. 23, lines 60-61; col. 25, lines 45-63; col. 34, lines 22-37; col. 35, lines 15-28; and col. 37, line 29, to col. 30, line 5).

[7] Levinson US-909 similarly notes the two pertinent Th cell types Th1 and Th2 and their functions, etc. (col. 1, lines 37-57; col. 2, lines 1-60; col. 3, lines 1-10; col. 7, lines 8-18; col. 36, lines 35-66; and col. 38, line 58, to col. 39, line 6).

[8] Levinson US-218 (138 columns with 37 sheets of drawings), which is a division of [13] Levinson US-371 and in turn a CIP of [10] Levinson US-351 which is itself a CIP of [5] Levinson US-322), insofar as pertinent hereto, similarly to [5] Levinson US-322, shows treatment of T lymphocyte related immune disorders, especially Th2 dominated disorders, including chronic inflammatory diseases and disorders such as Crohn's disease, reactive arthritis, including Lyme disease, insulin dependent diabetes, organ specific autoimmunity, including multiple sclerosis, Hashimoto's thyroiditis and Grave's disease, contact dermatitis, psoriasis, graph rejection, graph versus host disease, sarcoidosis, atopic conditions, such as asthma and allergy, including allergic rhinitis, gastrointestinal allergies, including food allergies, eosinophilia, conjunctivitis, glomerular nephritis, certain pathogen susceptibilities, such as helminthic (e.g., leishmaniasis), and certain viral infections, including HIV, and bacterial infections, including tuberculosis and lepromatous leprosy (col. 1, lines 16-34; col. 3, lines 28-40 and 46-61; col. 5, lines 45-67; col. 13, line 65, to

col. 14, line 4; col. 16, lines 16-23; col. 17, lines 15-29; col. 40, lines 50-67; col. 53, line 64, to col. 54, line 26; col. 72, lines 22-24 and 29-32; and col. 74, lines 33-46).

[8] Levinson US-218 similarly notes the two pertinent Th cell types Th1 and Th2 and their functions, etc. (col. 1, lines 52-56; col. 1, line 63 to col. 2, line 7; col. 2, lines 21-34; col. 2, line 38 to col. 3, line 25; col. 6, lines 36-39 and 55-61; col. 7, lines 8-25, 34-35 and 44-48; col. 8, lines 39-59; col. 8, line 64, to col. 9, line 8; col. 9, lines 19-23; col. 9, line 66, to col. 10, line 12; col. 16, lines 45-51; col. 16, line 64, to col. 17, line 11; col. 24, lines 4-6; col. 41, lines 49-52; col. 48, lines 37-39; col. 48, line 62, to col. 50, line 3; col. 53, lines 14-20 and 51-59; col. 55, lines 19-49; col. 65, lines 58-63; col. 66, lines 1-4 and 26-28; col. 67, lines 50-53; and col. 68, lines 14-18).

Like [4] DeKruyff US-898 and [5] Levinson US-322, the claims of [8] Levinson US-218 further confirm that current patent practice permits language (similar to that herein) as to a method for diagnosing a "Th cell subpopulation-related disorder" (claims 10, 17 and 18) such as a "Th1 cell subpopulation-related disorder" (claim 11), e.g., multiple sclerosis, psoriasis or insulin dependent diabetes (claim 12).

[9] Kingsbury US-334 (138 columns with 33 sheets of drawings), insofar as pertinent hereto, similarly to [5] Levinson US-322, shows treatment of T lymphocyte related immune disorders, especially Th2 dominated disorders, including chronic inflammatory diseases

and disorders such as Crohn's disease, reactive arthritis, including Lyme disease, insulin dependent diabetes, organ specific autoimmunity, including multiple sclerosis, Hashimoto's thyroiditis and Grave's disease, contact dermatitis, psoriasis, graph rejection, graph versus host disease, sarcoidosis, atopic conditions, such as asthma and allergy, including allergic rhinitis, gastrointestinal allergies, including food allergies, eosinophilia, conjunctivitis, glomerular nephritis, certain pathogen susceptibilities, such as helminthic (e.g., leishmaniasis), and certain viral infections, including HIV, and bacterial infections, including tuberculosis and lepromatous leprosy (col. 1, lines 10-25; col. 3, lines 32-48; col. 7, lines 36-58; col. 13, line 64, to col. 14, line 11; col. 48, lines 8-23; col. 52, lines 15-23 and 35-50; col. 73, lines 45-58; and col. 77, lines 9-49).

[9] Kingsbury US-334 similarly notes the two pertinent Th cell types Th1 and Th2 and their functions, etc. (col. 1, line 35, to col. 2, line 57; col. 3, lines 4-29; col. 3, line 52 to col. 4, line 20; col. 4, lines 43-52 and 60-64; col. 5, lines 1-41; col. 6, lines 1-11; col. 7, lines 61-62; col. 7, line 66, to col. 8, line 2; col. 8, lines 53-59; col. 8, line 65, to col. 9, line 13; col. 17, lines 39-65; col. 52, lines 57-64; col. 53, lines 15-20 and 29-34; col. 54, lines 15-30 and 42-48; and col. 63, lines 47-49).

Like [4] DeKruyff US-898, [5] Levinson US-322 and [8] Levinson US-218, the claims of [9] Kingsbury US-334 additionally confirm that current patent practice permits language (similar to that

herein) as to an isolated nucleic acid molecule "expressed specifically by Th2 cells" (claims 4 and 6).

As is clear from the instant disclosure, per the present invention, relaxin (RLX) has an effect on the differentiation of antigen-specific $CD4^+$ T cells into IFN-γ (Th1) and/or IL-4 (Th2) producing cells, and on the production of IFN-γ and IL-4 induced by T cell receptor (TCR) stimulation of established T cell clones, permitting development of antigen-specific $CD4^+$ T cells into T cells showing enhanced ability to produce IFN-γ (Th1) without exerting any effect on production of IL-4 (Th2) (spec., p. 3, lines 8-15; p. 4, lines 6-14; and p. 14, lines 1-4 and 6-12). Such promoting effect of relaxin on development of IFN-γ producing cells is not due to relaxin induced release of IL-12 and/or IFN-α by antigen presenting cells (APC) (spec., p. 3, line 24, to p. 4, line 2; p. 12, lines 1-10; and p. 12, lines 4-6).

Considering the instant disclosure in the light of the voluminous references discussed above, it is believed that the scope and import, i.e., metes and bounds, of treating a "Th2-dominated disease" and inhibiting a "pathogenic Th2 response" as used in the claims herein are acceptable as definite descriptive medical uses under the circumstances.

Withdrawal of the indefiniteness rejection under 35 USC 112, second paragraph, is accordingly urged.

II - DOUBLE PATENTING REJECTION

Reconsideration is requested of the obviousness-type non-statutory double patenting rejection of claims 1-5 herein as

unpatentable over claims 20-28 of Bigazzi US-296 in view of Piccinni et al., i.e., Annals of the New York Academy of Sciences: Neuroimmuno modulation 2000, 917: 844-852, "Environmental Factors Favoring the Allergen-specific Th2 response in Allergic Subjects," co-authored by Marie-Pierre Piccinni, Enrico Maggi and Sergio Romagnani ("Piccinni-844"), and which is a paper presented at the 4th International Congress of the International Society for Neuroimmuno modulation held on September 29-October 2, 1999 in Switzerland.

To expedite prosecution, the accompanying Terminal Disclaimer is presented as to Bigazzi US-296, i.e., related U.S. Patent No. 5,952,296, issued September 14, 1999 of common inventorship herewith.

Hence, the double patenting rejection is believed to be overcome and withdrawal thereof is respectfully requested.

CONCLUSION

In view of the foregoing, the present invention is believed to be patentable under 35 USC 102 and 35 USC 103 over the pertinent prior art, and the specification and claims are believed to be in permissible form and of adequately supported scope and import under 35 USC 112, first and second paragraphs.

Reconsideration and allowance are respectfully requested.

Favorable action is earnestly solicited.

Respectfully submitted
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Application Project

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<130> AppFileReference : 67206
<140> CurrentAppNumber : US 09/606,569
<141> CurrentFilingDate : 2000-06-29

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Custom Codon

Sequence Name : SEQ ID NO:1

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Application Project

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<141> CurrentFilingDate : 2000-06-29

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